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ACUTE NONSPECIFIC PERICARDITIS

The interest of clinicians in acute pericarditis among adult persons has been stimulated during the past ten years with the recognition of the characteristic electrocardiographic pattern and of the symptom complex in which pain may be the predominant feature. Although pericarditis has been recognized as a complication of pneumonia and septicemia or part of the clinical picture of rheumatic fever, tuberculosis, disseminated lupus erythematosis, myocardial infarction and uremia, there is a group of pericarditic patients in whom the etiologic factors are unknown or obscure.

This group of patients, who have a condition of undetermined etiology, is considered to be important because the clinical features are related mainly to the pericarditic inflammatory process, and the severe pain may simulate that arising from acute myocardial infarction.[1,2] The terminology best suited to this clinical syndrome is difficult to choose because of the undoubted variability in the causative agent and because the pericarditis probably is always secondary, even though it assumes a primary role in initiating the patient's complaints. Diagnostic phrases such as "acute nonsuppurative," "acute serofibrinous," "acute idiopathic," "primary idiopathic," "acute benign," and "acute nonspecific" pericarditis, have been used. The last term is the one that is preferred herein. It has been frequently customary to modify the diagnostic term by a phrase which indicates that the disease has a high incidence among young adult persons. Recent observations, however, have emphasized that the condition may occur in any decade of adult life, and that it is not limited to the younger age group. In a few patients who have had an attack of this type of pericarditis, recurrences of a similar benign type of pericarditis have been experienced, and for this syndome the term "acute relapsing pericarditis" may be suggested.

Clinical features

The patient usually dates the beginning of his illness to the onset of the thoracic pain, and the infection in the upper part of the respiratory tract which frequently precedes the condition may have been forgotten. The pain may begin with dramatic suddenness, with immediate incapacitation of the patient, or it may be mild and intermittent for several days before its greatest severity is attained. In rare instances after the onset of severe pain, there may be circulatory collapse and the patient may be observed to be in a state of clinical shock. The distribution of the pain may be widespread over the whole thorax, or localized in the substernal, precordial, epigastric or interscapular region. Shoulder tip pain may occur, and pain may extend into the arms. The pain may be described as "aching" or "squeezing," and is characteristically aggravated by breathing, coughing, twisting of the trunk and sometimes by swallowing. The patient may feel most comfortable in the sitting position. The pain in its severe form rarely lasts for more than a few hours and gradually becomes moderate over a period of two or three days. Some pain may persist in a mild form for many weeks, long after otherwise complete recovery.

Marked differences occur in the severity of the illness. In not a few cases, with the disappearance of pain in two or three days, the patient feels exceptionally well and convalescence is a matter of only a week or two. In the majority of cases, however, general malaise, fatigability and vague pains in the thorax persist for some weeks. In a few other patients, the fever may persist for two to three weeks and the ambulatory regime may be delayed, but convalescence may then be as rapid as in cases in which fever is of short duration.

In regard to the physical signs, the pericardial friction rub is by far the most important of these. If the patient has been under observation continuously throughout all phases of his illness, it is probable that this diagnostic sign will always be heard. Three patients have been seen who spontaneously gave the information that at the onset of their illness they were conscious of a friction rub within their thorax, correlated with the heart beat. The fever, which is usually maximal on the first day, rarely exceeds that indicated by a temperature of 102° F. (38.9° C.) and the accompanying tachycardia is slight or moderate. In one instance a clinical picture developed which suggested cardiac compression. The patient, a young soldier admitted to an overseas hospital with thoracic pain and in a state of shock, showed an increase of venous pressure in the midphase of illness. Pericardicentesis was carried out. The removal of a small amount of fluid had little effect on the clinical condition, but eventually convalescence was rapid and recovery complete.

Laboratory features

The laboratory observations in cases of acute nonspecific pericarditis reveal mild leukocytosis and an increased sedimentation rate, the former returning to normal within the first week and the latter within two weeks, as a rule.

Roentgenograms of the thorax made during the course of the acute pericarditic illness may be of great value because of changes in the size of the cardiac shadow. There can be no doubt that the cardiac silhouette may increase greatly in size when it is compared in teleroentgenograms, and this enlargement may appear as early as twelve hours after the onset of the pain. A certain degree of skepticism may be encountered in regard to the thesis that the increase in size represents actual cardiac dilatation and not pericardial effusion. Such a thesis, however, is accepted by many who have intensively studied the problem. In a few instances, the roentgenogram of the thorax made in the early phase of the illness will reveal mild pneumonitis, usually basal in distribution.

The electrocardiographic pattern has been described in detail by many authors and referred to by Barnes and me.[1] The essential components of the diagnostic pattern are the elevation of the RS-T segment with a dome-shaped or peaked T wave in the early stage, and negativity of the T waves in the late phase. There is no depression of the RS-T segment at any time; the T waves never show a classic reciprocal relationship in leads I and III, and no Q pattern ever develops in the standard or chest leads.

Differential diagnosis

When the diagnosis of acute pericarditis seems evident, the search for an etiologic factor should not be abandoned too quickly. Rheumatic fever and tuberculosis are two conditions that always have to be considered, but pericarditis due to these causes is more often painless and has a different clinical course.

As a general rule, the diagnostic problem will be in the distinction between the pain of pericarditis and that of acute myocardial ischemia or infarction. One of the main points in differential diagnosis is the aggravation of the pericarditic pain by breathing or body movements. Although the pain of pericarditis may extend into the neck and both shoulders, it usually will not spread down into the elbow and wrist joints. The presence of fever, a pericardial rub and an increased sedimentation rate on the first day of the pain, instead of after a period of some days, may be helpful in support of the diagnosis of pericarditis. The other conditions that may be considered in differential diagnosis are lesions in the posterior part of the mediastinum, such as mediastinitis, pneumomediastinum, diaphragmatic hernia, or aortic dissection. If the possibility of the presence of such conditions is kept in mind, they should not give trouble in differential diagnosis.

Prognosis

In spite of the fact that the pathologic processes may vary in different patients, the prognosis seems to be universally good. The condition of the fourteen patients reported on by Barnes and me has been closely followed, and nothing has been learned to change the opinion that acute pericarditis in adult persons usually is a benign type of disease without sequelae. There is now an additional group of twenty-nine patients whose condition has been diagnosed, since that time, by the cardiac consultants at the Mayo Clinic, as acute nonspecific pericarditis with pain, and the condition of these patients has been followed for periods of one year to five years. Further emphasis may be placed again on the recurrent vague precordial pains which may persist for weeks, long after the convalescence is otherwise complete. In such patients with persisting pains, particularly if the pains are associated with a prolonged asthenic state, functional symptoms may make their appearance and tax the physician's patience and skill in maintaining the patient's morale. At least four patients have had recurring attacks, with the electrocardiogram returning to normal or nearly so between the attacks. One patient, a physician, (case 5, Barnes' and my series) was, at the time of the present writing, enjoying a very active life and carrying on a busy consulting practice in spite of three recurrences.

Comment

Many aspects of the benign syndrome of acute pericarditis in adult persons lack adequate explanation. Among the problems presented are the etiologic agents responsible for the illness, the involved tissue within the thorax from which the pain stimulus originates, the factors responsible for the circulatory collapse that may occur with the onset of pain, the mechanism of cardiac dilatation, the reasons for the persistence of the pain and the nature of the electrophysiologic disturbance which gives rise to the electrocardiographic picture.

Eventually, it may be possible to reach a more clear-cut opinion as to the causes of the nonspecific pericardial inflammation, but at present it is assumed that the serous surfaces of the pericardium may respond similarly to a number of directly infectious agents or products of such agents. The high incidence of pericarditis of unknown etiology among adult persons has been noted by Nay and Boyer[3] and Smalley and Ruddock.[4]

The nature of the distribution of pain and the aggravation of the pain by breathing, swallowing and movements of the trunk suggest that the pain stimulus arises in the parietal pericardium and that it is more truly pleuropericardial than pericardial in origin. This concept is not only in conformance with the results of Capps's[5] observations on pericardial pain, but it explains why pericarditis, within the pericardium, may be painless. The severe circulatory collapse that may occur is associated with severe pain, and it might more readily be believed that the mechanism is analogous to the state of severe shock that may be observed in some cases of "chokes" in decompression sickness, and to spontaneous pneumomediastinum, rather than to some direct effect on the myocardium.

The mechanism of the cardiac dilatation is obscure, in spite of analyses of the possible causative factors. The possibilities of severe myocarditis, coronary insufficiency caused by arterial spasm, superficial myocardial edema limiting systole and the loss of the constraining influence of the pericardium, are imaginative factors, without basis in real evidence. It would be easier to assume pericardial effusion as a basis for the enlargement of the cardiac silhouette, but roentgenographically and clinically the evidence is against the diagnosis of pericardial effusion.

Acute pericarditis constitutes one of the best examples of how marked electrocardiographic changes may exist in the presence of relatively normal cardiac function and structure. The correlation between the electrocardiographic changes and subepicardial myocarditis has been emphasized, but actually such emphasis may mislead one into thinking too rigidly of structural changes rather than, biochemically, of cellular and membrane function. In other words, it is believed that surface injury might occur without much histologic aberration, and that the ingress of inflammatory cells in an area would not necessarily mean change in cellular depolarization and repolarization. The extent of segment elevation in the chest leads, and the position on the chest at which the maximal change occurs, are unpredictable. Two possible explanations for variation in the chest-lead patterns are, first, one's ignorance of the uniformity of the epicardial injury, and second, the fact that in some positions on the chest, anterior and posterior injury potentials may partly neutralize one another.

The points having been made that the pain is related to the exterior of the pericardium and the electrocardiographic changes to the epicardium, it might be expected that the two conditions would not necessarily parallel one another. Indeed, as is well known, marked electrocardiographic changes characteristic of pericarditis may exist without pain, and it should be expected occasionally that thoracic pain characteristic of pericarditis will be encountered without the electrocardiographic pattern of pericarditis. After the results of experimental work, there was some hope that it would be possible to localize pericarditis in some instances, and to correlate the electrocardiographic pattern with the distribution of pain. To date, there has been no case in which the diagnosis of localized pericarditis has been made, and there has been no correlation of the electrocardiographic pattern with the distribution of pain recognized.

Summary

The salient points in the problems of the diagnosis of acute pericarditis have been reviewed. Emphasis has been directed toward the clinical syndrome of severe pain and the electrocardiographic abnormalities.

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